MEDLINE

AN 1999008337 MEDLINE

DN 99008337 PubMed ID: 9794235

TI Expression of T:G mismatch-specific thymidine-DNA glycosylase and **DNA methyl transferase** genes during development and tumorigenesis.

AU Niederreither K; Harbers M; Chambon P; Dolle P

CS Institut de Genetique et de Biologie Moleculaire et Cellulaire, CNRS/INSERM/ULP/College de France, Illkirch, C.U. de Strasbourg.

SO ONCOGENE, (1998 Sep 24) 17 (12) 1577-85.
Journal code: 8711562. ISSN: 0950-9232.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199811

ED Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981112

AB In situ hybridization was used to characterize the expression pattern of the T:G mismatch-specific thymidine-DNA glycosylase (TDG) gene, encoding a DNA repair enzyme which corrects G:T mismatches that result from the hydrolytic deamination of 5-methyl cytosines. TDG transcripts were uniformly and ubiquitously expressed from 7.5-13.5 days post-coitum, but were then markedly enriched in specific tissues of the developing fetus. At 14.5 gestational days, TDG was strongly expressed in the developing nervous system, thymus, lung, liver, kidney and intestine. At later stages, high levels of expression were detected in the thymus, brain, nasal epithelium and within proliferating regions of the intestine, skin, kidney, teeth and bone. This pattern of expression strongly correlated with those of the methyl transferase (MTase) gene, coding for the enzyme which specifically methylates CpG dinucleotides, and the p53 tumour suppressor gene. However, TDG and MTase were differentially expressed during maturation of the male and female germline. We also report that tumors occuring in mice which overexpress MMTV-v-Ha-ras or MMTV-c-myc transgenes or mice heterozygous for p53 gene disruption, all show elevated TDG and MTase expression specific to the transformed tissue.

(FILE 'HOME' ENTERED AT 15:40:15 ON 28 MAR 2003)

FILE 'REGISTRY' ENTERED AT 15:41:07 ON 28 MAR 2003
L1 1 S GLIEKNIEL/SQEP
L2 1 S [GSC]LIEKNIEL/SQSP AND SQL=9
L3 1 S G[VIL]IEKNIEL/SQSP AND SQL=9
L4 1 S GL[VLI]EKNIE[LIE]/SQSP AND SQL=9

=> s l1 or l2 or l3 or l4
L5 1 L1 OR L2 OR L3 OR L4

=> d sqd,bib

L5 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN 477561-32-3 REGISTRY
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL 9

PATENT ANNOTATIONS (PNTE):

Sequence Source	Patent Reference
Not Given	WO2002094981
	claimed
	SEQID 13

SEQ 1 GLIEKNIEL
=========
HITS AT: 1-9

REFERENCE 1

AN 138:13498 CA
TI Method of identifying peptides capable of binding to MHC molecules for treating cancers and autoimmune diseases
IN Barnea, Eilon; Beer, Ilan; Ziv, Tamar; Admon, Arie; Dassau, Lior; Buchsbaum, Samuel
PA Technion Research and Development Foundation Ltd., Israel
SO PCT Int. Appl., 238 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002094981	A2	20021128	WO 2002-IL383	20020516
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	US 2001-290958P		20010516		
	US 2001-865548		20010529		

NEWS 46 Feb 26 NTIS now allows simultaneous left and right truncation
NEWS 47 Feb 26 PCTFULL now contains images
NEWS 48 Mar 04 SDI PACKAGE for monthly delivery of multifile SDI results
NEWS 49 Mar 19 APOLLIT offering free connect time in April 2003
NEWS 50 Mar 20 EVENTLINE will be removed from STN
NEWS 51 Mar 24 PATDPAFULL now available on STN
NEWS 52 Mar 24 Additional information for trade-named substances without structures available in REGISTRY
NEWS 53 Mar 24 Indexing from 1957 to 1966 added to records in CA/CAPLUS

NEWS EXPRESS January 6 CURRENT WINDOWS VERSION IS V6.01a, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 01 OCTOBER 2002
NEWS HOURS STN Operating Hours Plus Help Desk Availability
NEWS INTER General Internet Information
NEWS LOGIN Welcome Banner and News Items
NEWS PHONE Direct Dial and Telecommunication Network Access to STN
NEWS WWW CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 16:08:11 ON 28 MAR 2003

=> file biosis
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'BIOSIS' ENTERED AT 16:08:47 ON 28 MAR 2003
COPYRIGHT (C) 2003 BIOLOGICAL ABSTRACTS INC.(R)

FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 26 March 2003 (20030326/ED)

=> d his

(FILE 'HOME' ENTERED AT 16:08:11 ON 28 MAR 2003)

FILE 'BIOSIS' ENTERED AT 16:08:47 ON 28 MAR 2003

=> s DNA methyl transferase
691590 DNA
10617 DNAS
693590 DNA
(DNA OR DNAS)
242331 METHYL
139 METHYLS
242407 METHYL

(METHYL OR METHYLS)
65153 TRANSFERASE
5960 TRANSFERASES
68145 TRANSFERASE
(TRANSFERASE OR TRANSFERASES)
L1 117 DNA METHYL TRANSFERASE
(DNA(W)METHYL(W)TRANSFERASE)

=> d 110-117

L1 ANSWER 110 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1980:265010 BIOSIS
DN BA70:57506
TI **DNA METHYL TRANSFERASE** EC-2.1.1.37 FROM THE EUKARYOTE CHLAMYDOMONAS-REINHARDI.
AU SANO H; SAGER R
CS DIV. MOL. GENET., SIDNEY FARBER CANCER INST., 44 BINNEY ST., BOSTON, MASS. 02115, USA.
SO EUR J BIOCHEM, (1980) 105 (3), 471-480.
CODEN: EJBCAI. ISSN: 0014-2956.
FS BA; OLD
LA English

L1 ANSWER 111 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1979:62336 BIOSIS
DN BR17:2336
TI DIFFERENTIATION AND DNA METHYLATION IN MOUSE ERYTHRO LEUKEMIA CELLS.
AU WEICH N; SCHNEIDERMAN N; ACS G; CHRISTMAN J K
SO Fed. Proc., (1979) 38 (3 PART 1), 668.
CODEN: FEPRA7. ISSN: 0014-9446.
DT Conference
FS BR; OLD
LA Unavailable

L1 ANSWER 112 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1979:61381 BIOSIS
DN BR17:1381
TI **DNA METHYL TRANSFERASE** EC-2.1.1.37 FROM CHLAMYDOMONAS-REINHARDI.
AU SAGER R; SANO H
SO Fed. Proc., (1979) 38 (3 PART 1), 487.
CODEN: FEPRA7. ISSN: 0014-9446.
DT Conference
FS BR; OLD
LA Unavailable

L1 ANSWER 113 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1978:58651 BIOSIS
DN BR15:2151
TI SEQUENCE SPECIFICITY OF **DNA METHYL TRANSFERASE** OF MOUSE ERYTHRO LEUKEMIA CELLS.
AU PRICE P; VORSANGER G; SCHNEIDERMAN N; CHRISTMAN J K
SO Fed. Proc., (1978) 37 (6), 1415.
CODEN: FEPRA7. ISSN: 0014-9446.
DT Conference
FS BR; OLD
LA Unavailable

L1 ANSWER 114 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1977:38619 BIOSIS
DN BR13:38619
TI IN-VITRO STUDIES ON METHYLATION OF DNA IN RAT BRAIN NUCLEI.
AU QUICK D P
SO Fed. Proc., (1977) 36 (3), 907.

CODEN: FEPRA7. ISSN: 0014-9446.
DT Conference
FS BR; OLD
LA Unavailable

L1 ANSWER 115 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1974:19665 BIOSIS
DN BR10:19665
TI ENZYMIC METHYLATION OF NATURAL POLY NUCLEOTIDES.
AU KERR S J; BOREK E
SO BOYER, PAUL D. (ED.). THE ENZYMES, VOL. IX. GROUP TRANSFER. PART B. PHOSPHORYL TRANSFER, ONE-CARBON GROUP TRANSFER, GLYCOSYL TRANSFER, AMINO GROUP TRANSFER, OTHER TRANSFERASES. 3RD ED. XXII+586P. ILLUS. ACADEMIC PRESS: NEW YORK, N.Y., U.S.A.; LONDON, ENGLAND. (1973) 167-195.
FS BR; OLD
LA Unavailable

L1 ANSWER 116 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1973:104250 BIOSIS
DN BA55:4243
TI DEFICIENCY OF THE DNA OF MICROCOCCUS-RADIODURANS IN METHYL ADENINE AND METHYL CYTOSINE.
AU SCHEIN A; BERDAHL B J; LOW M; BOREK E
SO BIOCHIM BIOPHYS ACTA, (1972) 272 (3), 481-485.
CODEN: BBACAQ. ISSN: 0006-3002.
FS BA; OLD
LA Unavailable

L1 ANSWER 117 OF 117 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1972:194307 BIOSIS
DN BA54:24301
TI STUDIES ON THE MODE OF ACTION OF NALIDIXIC-ACID.
AU PEDRINI A M; GEROLDI D; SICCARDI A; FALASCHI A
SO EUR J BIOCHEM, (1972) 25 (2), 359-365.
CODEN: EJBCAI. ISSN: 0014-2956.
FS BA; OLD
LA Unavailable

=> file medline
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	41.44	41.65

FILE 'MEDLINE' ENTERED AT 16:28:58 ON 28 MAR 2003

FILE LAST UPDATED: 27 MAR 2003 (20030327/UP). FILE COVERS 1958 TO DATE.

On June 9, 2002, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2003 vocabulary. See http://www.nlm.nih.gov/mesh/summ2003.html for a description on changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s ((DNA methyl transferase) or MTDM)/ti
142025 DNA/TI
1163 DNAS/TI
143065 DNA/TI
((DNA OR DNAS)/TI)
26524 METHYL/TI
7 METHYLS/TI

```
          26530 METHYL/TI
                (METHYL OR METHYLS)/TI)
           7577 TRANSFERASE/TI
           1284 TRANSFERASES/TI
           8807 TRANSFERASE/TI
                (TRANSFERASE OR TRANSFERASES)/TI)
             11 DNA METHYL TRANSFERASE/TI
                (DNA(W)METHYL(W)TRANSFERASE)/TI)
              0 MTDM/TI
L2           11 ((DNA METHYL TRANSFERASE) OR MTDM)/TI

=> d bib, abs 1-11
```

L2 ANSWER 1 OF 11 MEDLINE
AN 2002686592 MEDLINE
DN 22334389 PubMed ID: 12449729
TI Glutathione S-transferase and O6-methylguanine **DNA methyl transferase** activities in patients with thyroid papillary carcinoma.
AU Dincer Yildiz; Akcay Tulay; Celebi Nilgun; Uslu Ilhami; Ozmen Ozlem; Hatemi Husrev
CS Department of Biochemistry, Cerrahpasa Medical School, Istanbul University, Istanbul 34300, Turkey.
SO CANCER INVESTIGATION, (2002) 20 (7-8) 965-71.
Journal code: 8307154. ISSN: 0735-7907.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200212
ED Entered STN: 20021214
Last Updated on STN: 20021217
Entered Medline: 20021212
AB Alkylating agents, which are metabolized by glutathione S-transferase (GST), have an important role in the etiology of cancer by forming mutagenic DNA adducts. Previous studies have shown that DNA repair protein, O6-methylguanine DNA methyltransferase, repairs these mutagenic DNA adducts and its activity is correlated with the resistance of human tumors to alkylating agent-based anti-cancer drugs. However, little is known about GST and O6-methylguanine DNA methyltransferase activities in patients with thyroid cancer in vivo. We measured the activities of GST and O6-methylguanine DNA methyltransferase in the leukocytes from patients with papillary thyroid carcinoma and healthy controls. The GST activity was significantly higher in men than in women, and it was negative correlated with age in men whereas it was unchanged in women in the control group. Both GST and O6-methylguanine DNA methyltransferase activities were significantly increased in the patient group. There were no age and sex-related changes in the O6-methylguanine DNA methyltransferase activity in both the control and patient groups. These results suggest that leukocyte GST and O6-methylguanine DNA methyltransferase activities were increased with thyroid cancer. This may be related to the resistance to chemotherapy exhibited by patients with thyroid cancer.

L2 ANSWER 2 OF 11 MEDLINE
AN 2002613415 MEDLINE
DN 22257582 PubMed ID: 12370764
TI O(6)-methylguanine-**DNA methyl transferase** gene expression and prognosis in breast carcinoma.
AU Cayre Anne; Penault-Llorca Frederique; De Latour Monique; Rolhion Christine; Feillel Viviane; Ferriere Jean-Pierre; Kwiatkowski Fabrice; Finat-Duclos Francoise; Verrelle Pierre
CS Department of Pathology, Centre Jean Perrin, BP 392, 63011 Clermont-Ferrand Cedex, France.. top@cjp.u-clermont1.fr

SO INTERNATIONAL JOURNAL OF ONCOLOGY, (2002 Nov) 21 (5) 1125-31.
Journal code: 9306042. ISSN: 1019-6439.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200303
ED Entered STN: 20021010
Last Updated on STN: 20030305
Entered Medline: 20030304
AB O(6)-methylguanine-DNA methyl transferase (MGMT) in human carcinomas has been associated with tumor resistance to alkylating agents. The aims of this study were: i) to correlate tumor MGMT expression and patient and tumor characteristics in malignant breast carcinomas treated with induction chemotherapy including cyclophosphamide (CPM) and ii) to study the predictive and prognostic values of tumor MGMT gene expression. We used RT-PCR to measure the levels of tumor MGMT expression in 107 patients with breast carcinomas prior to neoadjuvant chemotherapy. Sixty patients (56%) received anthracyclines and CPM and 47 (44%) received only anthracyclines. Low levels of MGMT expression correlated with Scarff-Bloom-Richardson grade III ($p<0.005$), elevated S-phase ($p<0.05$), negative estrogen receptors ($p<0.05$), metastatic status ($p<0.05$) and occurrence of death ($p=0.01$). MGMT expression was not predictive of treatment response. Unexpectedly, survival was longer when tumor MGMT expression was high ($p<0.005$). The 4-year survival rate was 76% for high level MGMT patients and only 55% for others. This difference is also significant using the COX model ($p<0.05$). In breast cancer, tumor MGMT expression was not predictive of response to CPM. A low MGMT expression was significantly related to poor survival.

L2 ANSWER 3 OF 11 MEDLINE
AN 2002188894 MEDLINE
DN 21919408 PubMed ID: 11922418
TI An approach to the evaluation of the activity of the DNA repair enzyme O6-methylguanine-**DNA-methyl-transferase** in tumor tissue in vivo: syntheses of 6-benzyloxy-9-(2-[18F]fluoroethyl)-9H-purin-2-yl-amine and 6-benzyloxy-7-(2-[18F]fluoroethyl)-7H-purin-2-yl-amine.
AU Schirrmacher Ralf; Nesseler Esther; Hamkens Wilhelm; Eichhorn Uta; Schreckenberger Mathias; Kaina Bernd; Rosch Frank
CS Institute of Nuclear Chemistry, Johannes Gutenberg-Universitat Mainz, Germany.
SO APPLIED RADIATION AND ISOTOPES, (2002 Mar) 56 (3) 511-7.
Journal code: 9306253. ISSN: 0969-8043.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200205
ED Entered STN: 20020403
Last Updated on STN: 20020602
Entered Medline: 20020531
AB The resistance of tumor cells to the cytostatic activity of methylating and chloroethylating anticancer drugs is determined by the level of expression of the DNA repair protein O6-methylguanine-DNA-methyl-transferase (MGMT). The synthesis of labelled 6-benzyloxy-9H-purin-2-ylamine derivatives should hence allow a quantification of the MGMT status of tumor and non-target tissue in vivo. 6-benzyloxy-9-(2-fluoroethyl)-9H-purin-2-yl-amine and 6-benzyloxy-7-(2-fluoroethyl)-7H-purin-2-yl-amine were synthesized and evaluated in vitro, both showing an affinity of 1.8 microM. 6-benzyloxy-9-(2-[18F]fluoroethyl)-9H-purin-2-yl-amine and 6-benzyloxy-7-(2-[18F]fluoroethyl)-7H-purin-2-yl-amine were synthesized by alkylation of 6-benzyloxy-9H-purin-2-ylamine with 1-[18F]fluoro-2-tosylethane in optimized yields of 41% and 20%, respectively.

Biodistribution studies were performed in nude mice, carrying mex+ (MGMT expressing) and mex- tumors.

L2 ANSWER 4 OF 11 MEDLINE
AN 2002111044 MEDLINE
DN 21819377 PubMed ID: 11805295
TI HemK, a class of protein methyl transferase with similarity to **DNA methyl transferases**, methylates polypeptide chain release factors, and hemK knockout induces defects in translational termination.
CM Comment in: Proc Natl Acad Sci U S A. 2002 Feb 5;99(3):1104-6
AU Nakahigashi Kenji; Kubo Naoko; Narita Shin-ichiro; Shimaoka Takeshi; Goto Simon; Oshima Taku; Mori Hirotada; Maeda Maki; Wada Chieko; Inokuchi Hachiro
CS Department of Biophysics, Graduate School of Science, Kyoto University, Sakyo-ku, Kyoto 606-8502, Japan.. knakahig@pobox.com
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Feb 5) 99 (3) 1473-8.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200203
ED Entered STN: 20020215
Last Updated on STN: 20030105
Entered Medline: 20020307
AB HemK, a universally conserved protein of unknown function, has high amino acid similarity with DNA-(adenine-N6) methyl transferases (MTases). A certain mutation in hemK gene rescues the photosensitive phenotype of a ferrochelatase-deficient (hemH) mutant in Escherichia coli. A hemK knockout strain of E. coli not only suffered severe growth defects, but also showed a global shift in gene expression to anaerobic respiration, as determined by microarray analysis, and this shift may lead to the abrogation of photosensitivity by reducing the oxidative stress. Suppressor mutations that abrogated the growth defects of the hemK knockout strain were isolated and shown to be caused by a threonine to alanine change at codon 246 of polypeptide chain release factor (RF) 2, indicating that hemK plays a role in translational termination. Consistent with such a role, the hemK knockout strain showed an enhanced rate of read-through of nonsense codons and induction of transfer-mRNA-mediated tagging of proteins within the cell. By analysis of the methylation of RF1 and RF2 in vivo and in vitro, we showed that HemK methylates RF1 and RF2 in vitro within the tryptic fragment containing the conserved GGQ motif, and that hemK is required for the methylation within the same fragment of, at least, RF1 in vivo. This is an example of a protein MTase containing the DNA MTase motif and also a protein-(glutamine-N5) MTase.

L2 ANSWER 5 OF 11 MEDLINE
AN 2000406895 MEDLINE
DN 20359694 PubMed ID: 10899996
TI m5C RNA and m5C **DNA methyl transferases** use different cysteine residues as catalysts.
AU Liu Y; Santi D V
CS Departments of Biochemistry and Biophysics, and Pharmaceutical Chemistry, University of California, San Francisco, CA 94143-0446, USA.
NC GM51232 (NIGMS)
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2000 Jul 18) 97 (15) 8263-5.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 200008
ED Entered STN: 20000901
Last Updated on STN: 20000901
Entered Medline: 20000824
AB A family of RNA m(5)C methyl transferases (MTases) containing over 55 members in eight subfamilies has been identified recently by an iterative search of the genomic sequence databases by using the known 16S rRNA m(5)C 967 MTase, Fmu, as an initial probe. The RNA m(5)C MTase family contained sequence motifs that were highly homologous to motifs in the DNA m(5)C MTases, including the ProCys sequence that contains the essential Cys catalyst of the functionally similar DNA-modifying enzymes; it was reasonable to assign the Cys nucleophile to be that in the conserved ProCys. The family also contained an additional conserved Cys residue that aligns with the nucleophilic catalyst in m(5)U54 tRNA MTase. Surprisingly, the mutant of the putative Cys catalyst in the ProCys sequence was active and formed a covalent complex with 5-fluorocytosine-containing RNA, whereas the mutant at the other conserved Cys was inactive and unable to form the complex. Thus, notwithstanding the highly homologous sequences and similar functions, the RNA m(5)C MTase uses a different Cys as a catalytic nucleophile than the DNA m(5)C MTases. The catalytic Cys seems to be determined, not by the target base that is modified, but by whether the substrate is DNA or RNA. The function of the conserved ProCys sequence in the RNA m(5)C MTases remains unknown.

L2 ANSWER 6 OF 11 MEDLINE
AN 2000071129 MEDLINE
DN 20071129 PubMed ID: 10602504
TI Expression of **DNA methyl-transferase** (DMT) and the cell cycle in human breast cancer cells.
AU Nass S J; Ferguson A T; El-Ashry D; Nelson W G; Davidson N E
CS Oncology Center, The Johns Hopkins University School of Medicine, 422 N. Bond Street, Baltimore, Maryland 21231, USA.
SO ONCOGENE, (1999 Dec 9) 18 (52) 7453-61.
Journal code: 8711562. ISSN: 0950-9232.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
ED Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000113
AB Estrogen receptor (ER)-negative breast cancer cells display extensive methylation of the ER gene CpG island and elevated DNA methyltransferase (DMT) expression compared to ER-positive cells. The present study demonstrates that DMT protein levels tightly correlate with S phase fraction in ER-positive cells, whereas ER-negative cells express DMT throughout the cell cycle. In addition, levels of p21CIP1, which disrupts DMT binding to PCNA, are inversely correlated with DMT levels. Therefore increased DMT expression in ER-negative cells is not simply due to elevated S-phase fraction, but rather to more complex changes that allow cells to escape normal cell cycle-dependent controls on DMT expression. Because ER-negative breast tumors often have activated growth factor pathways, the impact of these pathways on DMT expression was examined in ER-positive cells. Stable transfection with fibroblast growth factors (FGFs) 1 and 4 led to increased DMT expression that could not be accounted for by a shift in S phase fraction. Elevated DMT protein expression in FGF-transfectants was accompanied by a significant decrease in p21, again suggesting a reciprocal relationship between these two proteins. However, acquisition of an estrogen-independent phenotype, even in conjunction with elevated DMT levels, was not sufficient to promote ER gene silencing via methylation. These results indicate that multiple steps are required for de novo methylation of the ER CpG island.

L2 ANSWER 7 OF 11 MEDLINE
AN 1999008337 MEDLINE
DN 99008337 PubMed ID: 9794235
TI Expression of T:G mismatch-specific thymidine-DNA glycosylase and **DNA methyl transferase** genes during development and tumorigenesis.
AU Niederreither K; Harbers M; Chambon P; Dolle P
CS Institut de Genetique et de Biologie Moleculaire et Cellulaire, CNRS/INSERM/ULP/College de France, Illkirch, C.U. de Strasbourg.
SO ONCOGENE, (1998 Sep 24) 17 (12) 1577-85.
Journal code: 8711562. ISSN: 0950-9232.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199811
ED Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981112
AB In situ hybridization was used to characterize the expression pattern of the T:G mismatch-specific thymidine-DNA glycosylase (TDG) gene, encoding a DNA repair enzyme which corrects G:T mismatches that result from the hydrolytic deamination of 5-methyl cytosines. TDG transcripts were uniformly and ubiquitously expressed from 7.5-13.5 days post-coitum, but were then markedly enriched in specific tissues of the developing fetus. At 14.5 gestational days, TDG was strongly expressed in the developing nervous system, thymus, lung, liver, kidney and intestine. At later stages, high levels of expression were detected in the thymus, brain, nasal epithelium and within proliferating regions of the intestine, skin, kidney, teeth and bone. This pattern of expression strongly correlated with those of the methyl transferase (MTase) gene, coding for the enzyme which specifically methylates CpG dinucleotides, and the p53 tumour suppressor gene. However, TDG and MTase were differentially expressed during maturation of the male and female germline. We also report that tumors occuring in mice which overexpress MMTV-v-Ha-ras or MMTV-c-myc transgenes or mice heterozygous for p53 gene disruption, all show elevated TDG and MTase expression specific to the transformed tissue.

L2 ANSWER 8 OF 11 MEDLINE
AN 89051746 MEDLINE
DN 89051746 PubMed ID: 3191474
TI Effect of nutritional zinc-deficiency on O6-alkylguanine-**DNA-methyl-transferase** activities in rat tissues.
AU Fong L Y; Cheung T; Ho Y S
CS Department of Biochemistry, Faculty of Medicine, University of Hong Kong.
SO CANCER LETTERS, (1988 Nov) 42 (3) 217-23.
Journal code: 7600053. ISSN: 0304-3835.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198812
ED Entered STN: 19900308
Last Updated on STN: 19980206
Entered Medline: 19881228
AB The effect of nutritional zinc-deficiency on the activities of O6-alkylguanine:DNA methyltransferase (AGT) in 9 rat tissues including liver, lung, kidney, spleen, brain, esophagus, forestomach, gastric-stomach and small intestine has been examined. Individual tissue extracts prepared from zinc-deficient and pair-fed, zinc-sufficient rats were incubated with N-[3H]methylnitrosourea-methylated calf thymus DNA for 1 h. The activities of AGT in these tissues were measured by two methods: (a) the transfer of the methyl group from O6-methylguanine in substrate DNA to AGT protein, and (b) the determination of the ratio of

O6-methylguanine:7-methylguanine remaining in substrate DNA following incubation. AGT activities (expressed as fmol protein methylated/h per mg protein) were significantly reduced in the esophagus, spleen and lungs of zinc-deficient rats as compared to those in their corresponding zinc-sufficient counterparts. The ratio of O6-methylguanine:7-methylguanine was also reduced in the esophagus of the zinc-deficient rat. These results were consistent with our earlier findings that dietary zinc-deficiency enhances nitrosamine-induced esophageal carcinogenesis in rats.

L2 ANSWER 9 OF 11 MEDLINE
AN 88026693 MEDLINE
DN 88026693 PubMed ID: 3664453
TI Effect of the esophageal carcinogen methylbenzylnitrosamine and of a putative potentiating factor, a trichothecene mycotoxin, on O6-methylguanine-**dna methyl transferase** in rat esophagus and liver.
AU Craddock V M; Henderson A R
CS MRC Toxicology Unit, Carshalton, Surrey, U.K.
SO CANCER LETTERS, (1987 Oct) 37 (1) 81-6.
Journal code: 7600053. ISSN: 0304-3835.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198711
ED Entered STN: 19900305
Last Updated on STN: 19980206
Entered Medline: 19871123
AB Epidemiological evidence from China and South Africa has implicated Fusaria mycotoxins in the etiology of esophageal cancer, although treatment of animals with extracts of Fusaria cultures did not cause cancer of the esophagus. Fusaria are the major producers of trichothecenes, and animal experiments have shown that these mycotoxins can damage the esophagus but they have not been shown to cause esophageal cancer. A plausible concept is therefore that esophageal cancer is initiated by the potent environmental esophageal carcinogens, certain nitrosamines, but that the levels of exposure are too low to cause clinical cancer unless their effects are enhanced by additional risk factors. Among the most likely enhancing factors in the regions mentioned above are Fusaria mycotoxins. As trichothecenes are known to inhibit sulphydryl-dependent reactions and to inhibit protein synthesis, experiments were carried out to determine whether potentiation of cancer could be mediated via inhibition of the DNA repair protein O6-methylguanine-DNA methyl transferase (O6MG-MT). The effect of diacetoxyscirpenol (DS) on O6MG-MT was studied. Chronic or acute treatment with DS did not alter the level of O6MG-MT in esophagus, or affect the depletion which occurs after injection of methylbenzylnitrosamine, or alter the rate of reappearance of O6MG-MT. A high dose of DS induced O6MG-MT in liver. These results suggest that if trichothecenes are risk factors for esophageal cancer, the effect is unlikely to be mediated by inhibition of O6MG-MT. Induction of the repair protein in liver may be relevant in the animal toxicoses caused by consumption of trichothecenes, but is unlikely to be implicated in the etiology of liver cancer in man.

L2 ANSWER 10 OF 11 MEDLINE
AN 86278376 MEDLINE
DN 86278376 PubMed ID: 3733854
TI Effect of N-nitrosamines carcinogenic for oesophagus on O6-alkyl-guanine-**DNA-methyl transferase** in rat oesophagus and liver.
AU Craddock V M; Henderson A R
SO JOURNAL OF CANCER RESEARCH AND CLINICAL ONCOLOGY, (1986) 111 (3) 229-36.
Journal code: 7902060. ISSN: 0171-5216.

CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198609
ED Entered STN: 19900321
Last Updated on STN: 20000303
Entered Medline: 19860917
AB Several O6-alkylGua adducts have been shown to be removed from DNA during its repair by transfer of the alkyl group to a cysteine residue in a specific AAP, with the formation of S-alkylcysteine. As the reaction is stoichiometric and irreversible, the AAP content of the cell can be reduced or depleted. In vivo depletion by a high dose of nitrosamine can be used to test for the formation of a repairable alkylation adduct at the O6-position of guanine. In addition, if the carcinogenic potency of a nitroso compound for a particular organ is related to the persistence of the adduct in DNA, potency would depend not on the level of alkylation attained after treatment, but on whether this was sufficient to deplete the AAP content of the organ concerned and so to slow down repair, i.e. depletion of AAP is a more relevant estimate of potency than is the initial extent of DNA alkylation. Dose-response studies on target and non-target organs showed that depletion of AAP correlated with organotropy for those nitrosamines known to methylate DNA, i.e. with NDMA for liver, and with NMBzA for oesophagus. With NDEA, the results supported the suggestion that other adducts in addition to O6-alkylGua may be involved. NMPhA, an oesophageal specific carcinogen, did not deplete AAP in oesophagus, and induced AAP in liver. This result adds to the evidence that NMPhA does not alkylate DNA.

L2 ANSWER 11 OF 11 MEDLINE
AN 66124278 MEDLINE
DN 66124278 PubMed ID: 4286564
TI A lipopolysaccharide inhibitor of a **DNA methyl transferase**.
AU Falaschi A; Kornberg A
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1965 Dec) 54 (6) 1713-20.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196607
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19660719